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<input type="checkbox"/>	L16	(glutamine near derivative) and ((diabetic adj neuropath\$) or glomeruloscleros\$ or glomerulopath\$ or ((chronic or persistent) adj (kidney or renal) adj disease))	5
<input type="checkbox"/>	L15	l11 and ((gamma adj2 glutamyltransferase) or \$glutamyltransferase or (alpha adj2 glutamyltraspeptidase) or \$glutamyltraspeptidase or (gamma adj2 glutamyl adj (peptidyltransferase or transpeptidase)) or (\$glutamyl adj (peptidyltransferase or transpeptidase)) or (gamma adj glutamyltransferase) or \$glutamyltrasferase or (gamma adj gpt) or \$gpt or (gamma adj gt) or \$gt or (gamma adj gtp) or \$gtp)	333
<input type="checkbox"/>	L14	l11 and ((gamma adj2 glutamyltransferase) or \$glutamyltransferase or (alpha adj2 glutamyltraspeptidase) or \$glutamyltraspeptidase or (gamma adj2 glutamyl adj (peptidyltransferase or transpeptidase)) or (\$glutamyl adj (peptidyltransferase or transpeptidase)) or (gamma adj glutamyltransferase) or \$glutamyltrasferase or (gamma adj gpt) or \$gpt or (gamma adj gt) or \$gt or (gamma adj gtp) or \$gtp)	333
<input type="checkbox"/>	L13	L12 and l11	31
<input type="checkbox"/>	L12	((diabetic adj neuropath\$) or glomeruloscleros\$ or glomerulopath\$ or ((chronic or persistent) adj (kidney or renal) adj disease))	7335
<input type="checkbox"/>	L11	((At adj 125) or ACV or acivicin or (isoxazoleacetic adj acid))	1993
	<i>DB=PGPB; PLUR=YES; OP=OR</i>		
<input type="checkbox"/>	L10	L8 and ((diabetic adj neuropath\$) or glomeruloscleros\$ or glomerulopath\$ or ((chronic or persistent) adj (kidney or renal) adj disease))	0
	<i>DB=USPT; PLUR=YES; OP=OR</i>		
<input type="checkbox"/>	L9	L8 and ((diabetic adj neuropath\$) or glomeruloscleros\$ or glomerulopath\$ or ((chronic or persistent) adj (kidney or renal) adj disease))	19
<input type="checkbox"/>	L8	l7 or l4	315
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<input type="checkbox"/>	L6	L4 and (kidney or renal)	131
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Focal Segmental Glomerulosclerosis

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Focal Segmental Glomerulosclerosis

- One segment of one glomerulus is sclerosed (arrow).
- The other glomeruli are unremarkable.
- With the trichrome stain, collagenous tissues take on a blue color.

(Description By:H. Yamase, M.D.)

(Image Contrib. by: UCHC)

Focal Segmental Glomerulosclerosis

Etiology

- Focal segmental glomerulosclerosis** is not one disease but a histologic pattern of injury seen on light microscopy in patients with nephrotic range proteinuria.
- Focal segmental glomerulosclerosis** may be idiopathic or may be seen as a consequence of reflux nephropathy, AIDS nephropathy; heroin/drug addiction, or compensatory hypertrophy/hyperfiltration.

Pathogenesis

- Since **focal segmental glomerulosclerosis** is not a disease so much as a histologic consequence of several diseases processes, the initiating pathogenetic mechanisms are varied. The common element to cases of **focal segmental glomerulosclerosis** is injury manifested by sclerosis in the setting of nephrotic range proteinuria.,

Epidemiology

- Focal segmental glomerulosclerosis** is not one disease but a histologic pattern of injury seen on light microscopy in patients with nephrotic range proteinuria.
- Focal segmental glomerulosclerosis** may be idiopathic or may be seen as a consequence of reflux nephropathy, AIDS nephropathy; heroin/drug addiction, or compensatory hypertrophy/hyperfiltration.
- Immunogenetics: DR4.

General Gross Description

- The kidneys in **focal segmental glomerulosclerosis** would show no gross abnormalities in the early phases of the disease.
- As the disease progresses and more glomeruli become sclerosed, the parenchymal mass will decrease and the kidneys would appear small and shrunken.

General Microscopic Description

- The histologic recognition of the disease is dependent on examining a number of glomeruli together.
- Some of the glomeruli are normal and some show segmental sclerotic lesions.
- The sclerotic lesions show solidification of segments of the tuft and is composed of collapsed capillary basement membranes, increased collagenous matrix and few if any viable endothelial and mesangial cells.
- Some of the affected segments may show large hyaline deposits occluding previously patent capillary lumens.
- Direct immunofluorescence studies show IgM and C3 in the affected sclerosed segments.
- Electron microscopy shows collapsed basement membranes, excess mesangial matrix material and the hyaline deposits noted on light microscopy correspond to large electron dense material occupying the capillary luminal space, which may represent entrapped serum proteins.
- Occasional foam cells may be encountered in the sclerosed segments.
- The tubular compartment will show tracts of atrophic tubules, paralleling the degree of **glomerulosclerosis**.

Clinical Correlation

- Patients with **focal segmental glomerulosclerosis** manifest heavy proteinuria (3.5 gm or more per 24 hours) and the consequence of protein loss (hypoalbuminemia, hyperlipidemia, edema).
- Additional complications of heavy proteinuria include increased vulnerability to infections, increased risk of thrombotic and thromboembolic diseases.
- Depending on the stage of the disease, patients may show normal renal function, varying degrees of renal insufficiency, or renal failure.

References

- Cotran RS, Kumar V, Robbins SL: Robbins Pathologic Basis of Disease. 5th ed. Philadelphia, W.B. Saunders, 1994, pp. 952-954.
- Rose B. Renal Pathophysiology the essentials. Baltimore: Williams and Wilkins. 1994. Ch. 9.

Focal Segmental Glomerulosclerosis

Synopsis by: Harold Yamase M.D. (T71200M00003)[212]

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